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ADRENERGIC CONTROL OF SWIMBLADDER PERFUSION IN THE EUROPEAN EEL *ANGUILLA ANGUILLA*

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Summary

Adrenergic control of swimbladder blood flow was analysed in blood-perfused preparations of the European eel as well as *in situ* by recording the changes in swimbladder blood flow and blood pressure following an injection of catecholamine into the dorsal artery.

In blood-perfused swimbladder preparations, injection of the α-adrenergic agonist phenylephrine into the perfusion loop caused a marked dose-dependent increase in perfusion pressure at constant flow, while injection of the β-agonist isoproterenol slightly decreased perfusion pressure. The β-effect was not as pronounced as the α-adrenergic vasoconstriction and was observed only during the first application of catecholamine in each preparation.

In situ injection of adrenaline (final concentration 10^{-8}–10^{-9} mol kg^{-1} body mass) into the dorsal aorta caused a dose-dependent transient increase in dorsal aortic blood pressure and in cardiac output which, after 5–10 min, returned to resting levels. Swimbladder perfusion also increased initially after an injection of adrenaline, but after about 1–2 min suddenly decreased and then slowly recovered to preinjection levels. Following the injection of adrenaline into the dorsal aorta, blood pressure changes in vessels at the swimbladder pole of the rete mirabile revealed a similar biphasic pattern with an initial increase, a subsequent decrease and a slow return to preinjection levels, while pressure in the arterial influx vessel of the rete resembled dorsal aortic pressure.

After injection of the β-blocker propranolol, adrenaline evoked a smaller initial increase in blood flow, but the subsequent reduction in flow was even more pronounced. Injection, in addition, of the α-adrenergic blocker phentolamine abolished the sharp adrenaline-induced decrease in swimbladder perfusion.

It is concluded that α- and β-adrenergically controlled resistance vessels are located close to the rete mirabile, probably at the arterial entrance into the rete. These vessels control perfusion of the rete mirabile and of the swimbladder and are thus involved in the control of gas deposition into the swimbladder.

Introduction

Many fish possess a gas-filled swimbladder as a hydrostatic organ. To retain neutral

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buoyancy during vertical migrations in the face of varying hydrostatic pressure, the swimbladder volume must be kept constant by depositing gas when swimming down and by resorbing gas when ascending. Thus, to maintain neutral buoyancy, a sophisticated control system is necessary. Stretch receptors provide afferent information from the bladder wall (Qutob, 1962; Tytler and Blaxter, 1973). The efferent loop consists of at least two separate elements: control of the connection between the resorbing and the secretory part of the bladder, i.e. control of gas resorption, and control of the secretory activity of the bladder.

Opening and closing of the connection between the secretory and the resorbing part of a bladder appear to be under nervous control. In *Gadus morhua* and *Pollachius virens*, vagal stimulation caused opening of the oval, probably due to β-adrenergic relaxation of circular muscles and α-adrenergic contraction of radial muscles. Application of acetylcholine resulted in a contraction of the circular muscles, but this appears not to be mediated by cholinergic nerves (Nilsson, 1971; Ross, 1978), and Nilsson (1971) suggested that closure of the oval may occur because of a lack of opening impulses.

The secretory activity of the bladder can be modified by a change in acid production and acid release by the gas gland cells (Pelster and Scheid, 1992*b*). The acid is responsible for the initiation of the so-called single concentrating effect, the first step in gas deposition. A decrease in the single concentrating effect will reduce the gas partial pressure increase and thus the partial pressure gradient towards the swimbladder lumen, reducing the rate of gas deposition (Fänge, 1983; Pelster and Scheid, 1992*a*). The rate of gas deposition may also depend on swimbladder blood perfusion. Recent studies have revealed a significant correlation between the rate of gas deposition into the swimbladder and the blood perfusion of the bladder. Furthermore, under hypoxic conditions both were reduced, indicating that the regulation of swimbladder perfusion is involved in the regulation of the rate of gas deposition (Pelster and Scheid, 1992*b*).

Catecholamines may be important for the control of swimbladder perfusion, as a vasoconstriction in swimbladder vessels has been observed during stimulation of the splanchnic and vagus nerves, mediated by α-adrenergic receptors. Injection of catecholamines, however, did not affect the rate of gas deposition (Stray-Pedersen, 1970; Nilsson, 1972). Vasodilator nerves have not been described in the swimbladder (Fänge, 1983).

Compared with that of other organs, the swimbladder circulatory system is rather complex in that three capillary sections are arranged in series: the arterial capillaries of the rete mirabile, the capillary network of the swimbladder epithelium and the venous capillaries of the rete mirabile. This complex structure also raises the question of where a possible control site might be located. In the present study, the influence of catecholamines on swimbladder perfusion and on blood pressure in arterial and venous vessels proximal and distal to the rete mirabile has been analysed *in situ* and in blood-perfused preparations of the swimbladder of immobilized European eels, *Anguilla anguilla*. The results indicate that swimbladder perfusion can be regulated independently of the central systemic circulation as a result of the presence of α- and β-adrenergically controlled resistance vessels located in the arterial vessels of the rete mirabile.

Materials and methods

Specimens of the European eel *Anguilla anguilla* (body mass 350–600 g) were obtained from a local supplier and kept in a freshwater aquarium with aerated tap water at 12–16 ˚C until used for experiments. All experiments were performed at a room temperature of 20–22˚C.

Animal preparation and the apparatus for in situ *experiments*

Under anaesthesia, animals were quickly immobilized by penetrating the skull with a thick needle and by spinal pithing using a long wire, as described by Pelster and Scheid (1992*b*). The animals were placed into an eel holder, and the gills were irrigated with well-aerated tapwater at a flow rate of 1.5–2.0 l min^{-1}. The swimbladder was carefully exposed from the ventral side and freed of connective tissue. The connection between the secretory and the resorbing parts of the swimbladder was ligated between the two retia mirabilia. Blood vessels from other tissues entering the vein leaving the retia were also ligated. A catheter was inserted into the swimbladder for gas sampling and, after removal of swimbladder gas, the bladder was reinflated with 500 μl of gas to prevent the swimbladder walls from sticking together.

To analyse the effects of catecholamines and specific adrenoceptor antagonists on the swimbladder circulation, blood flow and blood pressure were recorded *in situ* with a largely undisturbed circulatory system. The dorsal artery and the swimbladder vein were non-occlusively cannulated for measurement of blood pressure. Each cannula was connected to a pressure transducer for blood pressure recording (Gould, Statham, BD 23 ID). Blood pressure in swimbladder vessels (at the swimbladder pole) was measured using a Servo-null micropressure system (model 900, World Precision Instruments, New Haven, Connecticut, USA), as used by Pelster and Burggren (1991). A cuff-type Doppler flow probe (0.5–1 mm i.d.) was placed around the artery supplying the retia after it had been carefully separated from the rete effluent vein. The transducer crystal of the flow probe was connected to a Doppler flowmeter (Bioengineering, Iowa, USA). The signal of the cuff-type flow probe was calibrated *in situ* at the end of each experiment. A PE 20 catheter filled with heparinized (200 i.u. ml^{-1}) saline was inserted occlusively into the artery supplying the swimbladder tissue. This catheter and the dorsal arterial catheter were connected to a peristaltic pump, and the bladder was perfused with blood drawn from the dorsal artery of the experimental animal. In some preparations, the bulbus arteriosus was exposed and a Doppler transducer crystal was implanted for measurement of cardiac output (uncalibrated recording). The general anatomy of the swimbladder, including the terminology used for the swimbladder blood vessels, the location of the flow probe and the various sites for measurement of blood pressure, is shown in Fig. 1. Blood velocity and all blood pressure signals were sampled at 20 Hz by a computer during the entire experiment using the software package BrainWave (Broomfield, Colorado).

Perfused swimbladder preparations

To measure the influence of catecholamines on the perfusion pressure of an artificially perfused swimbladder preparation, the dorsal artery was cannulated non-occlusively. The

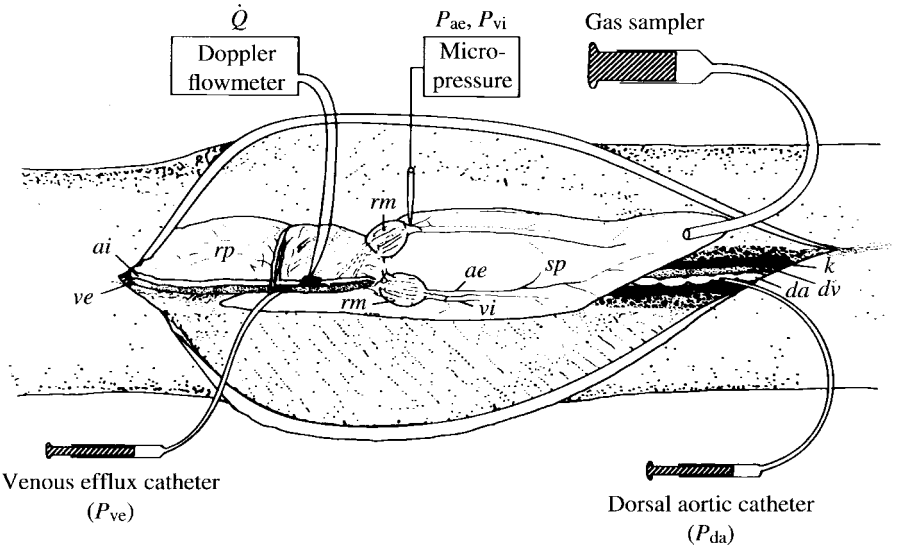


Fig. 1. Schematic drawing of the eel swimbladder showing the location of the flow probe and of the various catheters. *ai*, arterial influx; *ae*, arterial efflux of the rete; *da*, dorsal artery; *dv*, dorsal vein; *k*, kidney; *rm*, rete mirabile; *rp*, resorbing part of the swimbladder; *sp*, secretory part of the swimbladder; *ve*, venous efflux; *vi*, venous influx of the rete.

swimbladder artery was occlusively cannulated (PE 20). Using a peristaltic pump, blood was drawn from the dorsal artery at a flow rate of 0.1–0.4 ml min^{-1} (about 2–8 % of cardiac output) and directed into the swimbladder artery. The blood volume within the catheter loop amounted to about 0.8 ml. Thus, the swimbladder was perfused at a constant flow rate, and the perfusion pressure was measured by connecting the catheter to a pressure transducer (Gould, Statham, BD 23 ID).

Injection of catecholamines

Catecholamines were administered as bolus injections into the catheter loop in the perfused preparations. In the *in situ* studies, catecholamines that are present in the blood *in vivo*, i.e. adrenaline and noradrenaline, were injected into the dorsal aorta. The catheter was located distal to the origin of the swimbladder artery (Fig. 1), so that the bolus had to pass through the systemic circulation before it entered the swimbladder blood vessels. Catecholamines were injected as a bolus of 50–300 μl of a stock solution (10–100 μmol l^{-1}). Assuming full equilibration of the animal of 350–600 g, the final concentration in the animal would be about $10^{-8}-10^{-9}$ mol kg^{-1}, but the free concentration will probably be even lower owing to the concentration of amines in adrenergic nerves. The α- and β-adrenergic antagonists, phentolamine and propranolol respectively, were administered *in situ* in a bolus of 100 μmol l^{-1} stock solution, resulting in a concentration of 60 nmol kg^{-1} body mass. Stock solutions were prepared before starting the injections. All chemicals were obtained from Sigma.

Data analysis

For evaluation of the resistance of the various sections of the swimbladder circulation, a period of 3–5 min with stable blood pressure and blood flow recordings was analysed. Mean blood pressure and flow were obtained by averaging the original measurements. In respect of the blood pressure, three sections in the swimbladder circulation can be discriminated: the arterial capillaries of the rete mirabile (Ra, between ai and ae), the blood vessel network of the secretory part of the swimbladder (Sb, between ae and vi), and the venous capillaries of the rete mirabile (Rv, between vi and ve). The resistances (R) of these sections were calculated as:

$$R_{\mathrm{Ra}} = (P_{\mathrm{ai}} - P_{\mathrm{ae}})/\dot{Q}_{\mathrm{Sb}},$$

$$R_{\mathrm{Sb}} = (P_{\mathrm{ae}} - P_{\mathrm{vi}})/\dot{Q}_{\mathrm{Sb}},$$

$$R_{\mathrm{Rv}} = (P_{\mathrm{vi}} - P_{\mathrm{ve}})/\dot{Q}_{\mathrm{Sb}},$$

where P is pressure and $\dot{Q}$ is flow.

This calculation assumes that arterial blood flow equals venous flow, because only arterial flow has been measured. This assumption, however, appears reasonable as no secondary circulation has been described for the swimbladder; furthermore no blood vessels entering or leaving the secretory part of the swimbladder and bypassing the rete mirabile have been found. Unequal flow could also result from a water shift in the rete mirabile but, on the basis of measurements of haemoglobin concentration in blood vessels proximal and distal to the rete mirabile, Kobayashi *et al.* (1989) concluded that no significant water shift occurred between the arterial and venous vessels of the rete. Furthermore, model calculations have shown that equal blood flow in arterial and venous capillaries of the rete is crucial for the efficiency of the counter-current exchange system of the rete. A mismatch of only 3–5 % will virtually abolish the concentrating ability of the rete (Kobayashi *et al.* 1989).

Statistically significant differences in the observations were evaluated using the Wilcoxon signed-rank test or by one-way analysis of variance (ANOVA), followed by a multiple-comparison procedure (Bonferroni; SigmaStat). Significance of differences was accepted when $P<0.05$. Data are presented as mean ± S.E.M.

Results

Adrenergic effects on the swimbladder circulatory system independent of the central circulatory system have been tested by perfusing the swimbladder at constant flow with blood drawn from the dorsal artery, using a peristaltic pump. Catecholamines were dissolved in saline and injection of saline (sham injection) into the perfusion loop caused a transient decrease in perfusion pressure due to the transient decrease in haematocrit (Fig. 2A). Injection of the α-agonist phenylephrine into the perfusion loop, however, resulted in a marked transient increase in perfusion pressure (Fig. 2B). Injection of the β-agonist isoproterenol into the perfusion loop caused a slight decrease in perfusion pressure (Fig. 2C), while injection of the β-antagonist propranolol caused a transient increase in perfusion pressure (data not shown). For both drugs, the effect of a second

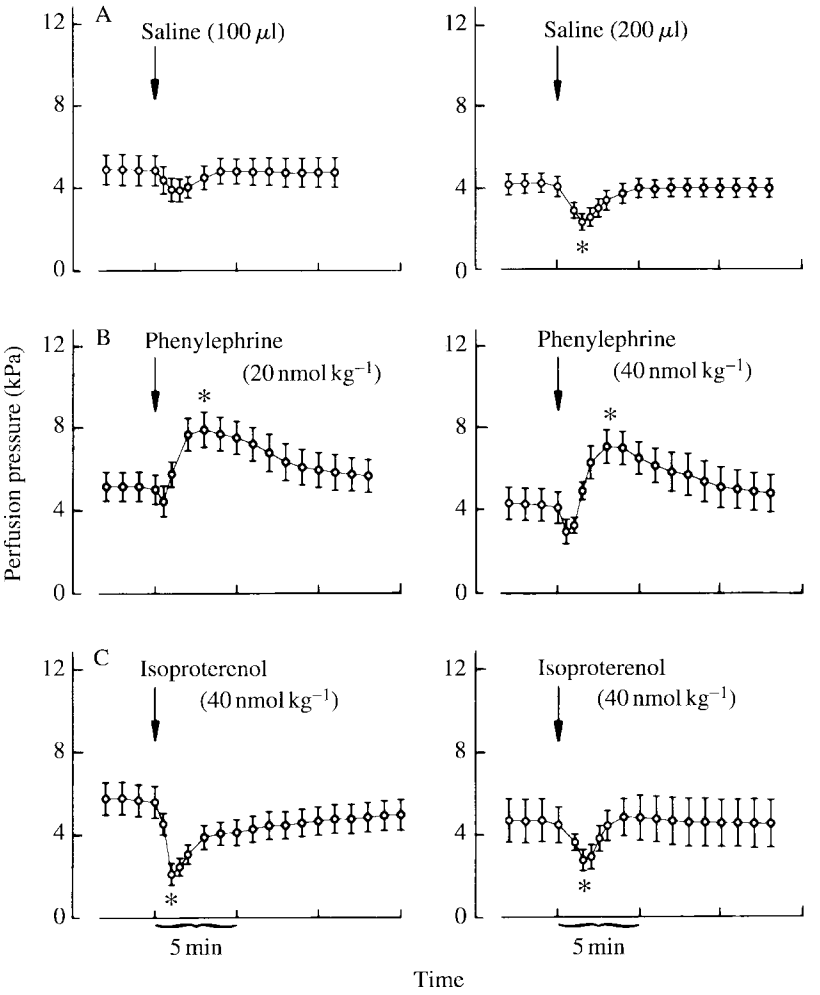


Fig. 2. Changes in perfusion pressure of blood-perfused swimbladder preparations (perfusion rate 0.3 ml min^{-1}) recorded during sham injection (saline) (A) and injection of adrenergic agonists (B,C) into the perfusion loop; values are mean ± S.E.M., N=6. Asterisks indicate significant differences compared with control values before injection of catecholamine.

injection about 20–30 min after the first one was much smaller than the effect of the first application and hardly differed from the effect of the sham injection.

The influence of catecholamines on the swimbladder circulatory system *in situ* has been analysed using bolus injections of the catecholamines found in the blood, i.e. adrenaline and noradrenaline, into the dorsal artery. Injection of adrenaline or noradrenaline into the dorsal aorta of the immobilized eel *in situ* resulted in a transient dose-dependent increase in cardiac output ($\dot{Q}_{tot}$) and in dorsal arterial blood pressure (P_{sys}). Within about 10 min after the injection, both variables had returned to the preinjection level (Fig. 3A,B). Swimbladder blood flow ($\dot{Q}_{Sb}$) also increased initially

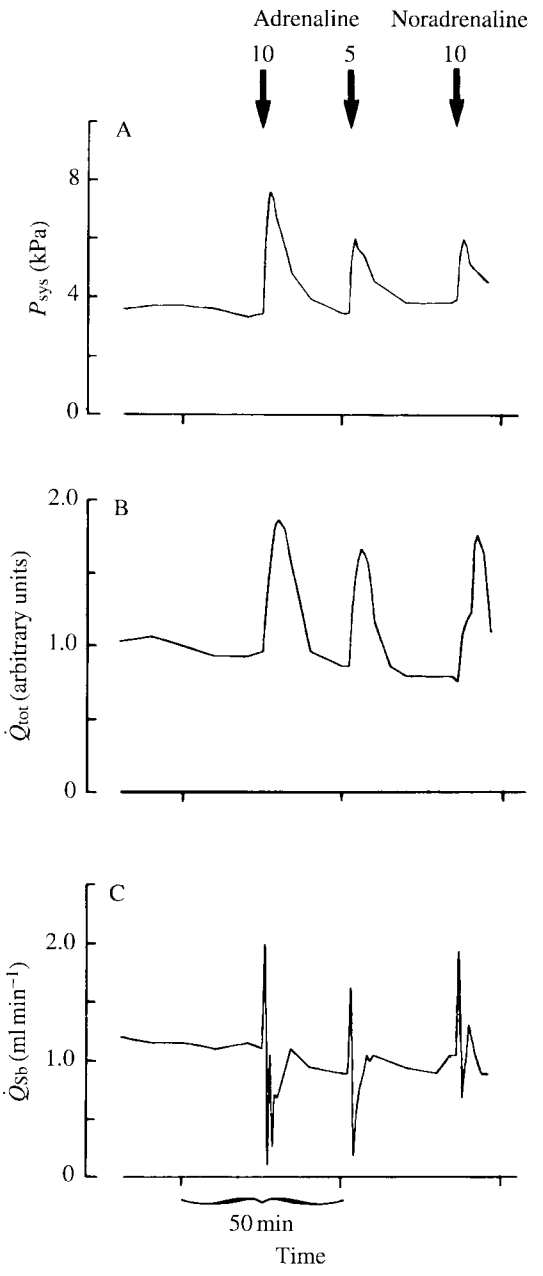


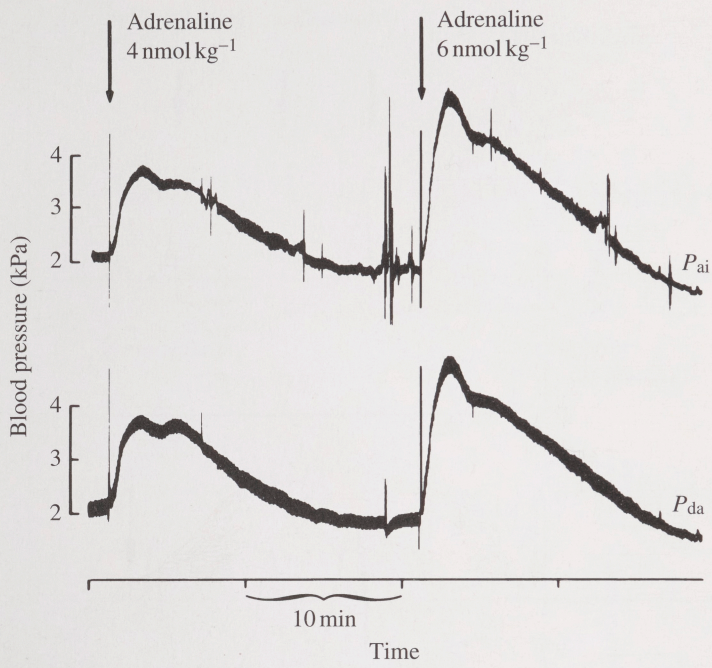
Fig. 3. Changes in dorsal arterial systolic blood pressure, P_{sys} (A), in cardiac output, $\dot{Q}_{tot}$ (B) and in swimbladder perfusion, $\dot{Q}_{Sb}$ (C) following bolus injections of catecholamines into the dorsal artery. First injection, 10 nmol kg^{-1} adrenaline; second injection, 5 nmol kg^{-1} adrenaline; third injection, 10 nmol kg^{-1} noradrenaline.

Fig. 4. Comparison of blood pressure changes during injections of adrenaline into the dorsal artery (da), measured with a conventional pressure transducer, and in the arterial influx into the rete mirabile (ai), measured with the micropressure system.

after an injection of catecholamine, but then, in spite of the still elevated cardiac output, decreased sharply and later slowly returned to preinjection levels (Fig. 3C).

Blood pressure recorded in the dorsal aorta with a conventional pressure transducer was nearly identical with blood pressure in the swimbladder artery (ai) recorded with the micropressure system. Blood pressure in these two blood vessels following catecholamine injections changed in parallel (Fig. 4). Typical pressure traces recorded in the arterial and venous blood vessels proximal and distal to the rete mirabile are presented in Fig. 5 (data from various preparations). In contrast to the arterial influx of the rete, where the pressure changes following an injection of adrenaline exactly follow the changes observed in the dorsal artery (see Fig. 4), blood pressure in the arterial efflux of the rete and in the venous influx of the rete follow a biphasic pattern with an initial rise followed by a sharp decrease. This biphasic pattern is similar to that of swimbladder blood flow after application of adrenaline (see Fig. 3C). In the venous efflux of the rete, the amplitudes of the biphasic pressure changes were very much reduced, but the general pattern was similar to that in the venous influx (vi) and arterial efflux (ae).

Mean values for blood pressure during control conditions measured in arterial and venous blood vessels proximal and distal to the rete mirabile reveal that the pressure drop between the arterial influx (ai) and efflux (ae) of the rete clearly exceeds the decrease between the venous influx and efflux of the rete and the decrease between the arterial efflux and venous influx of the rete (Fig. 6A). Accordingly, the resistance of the arterial



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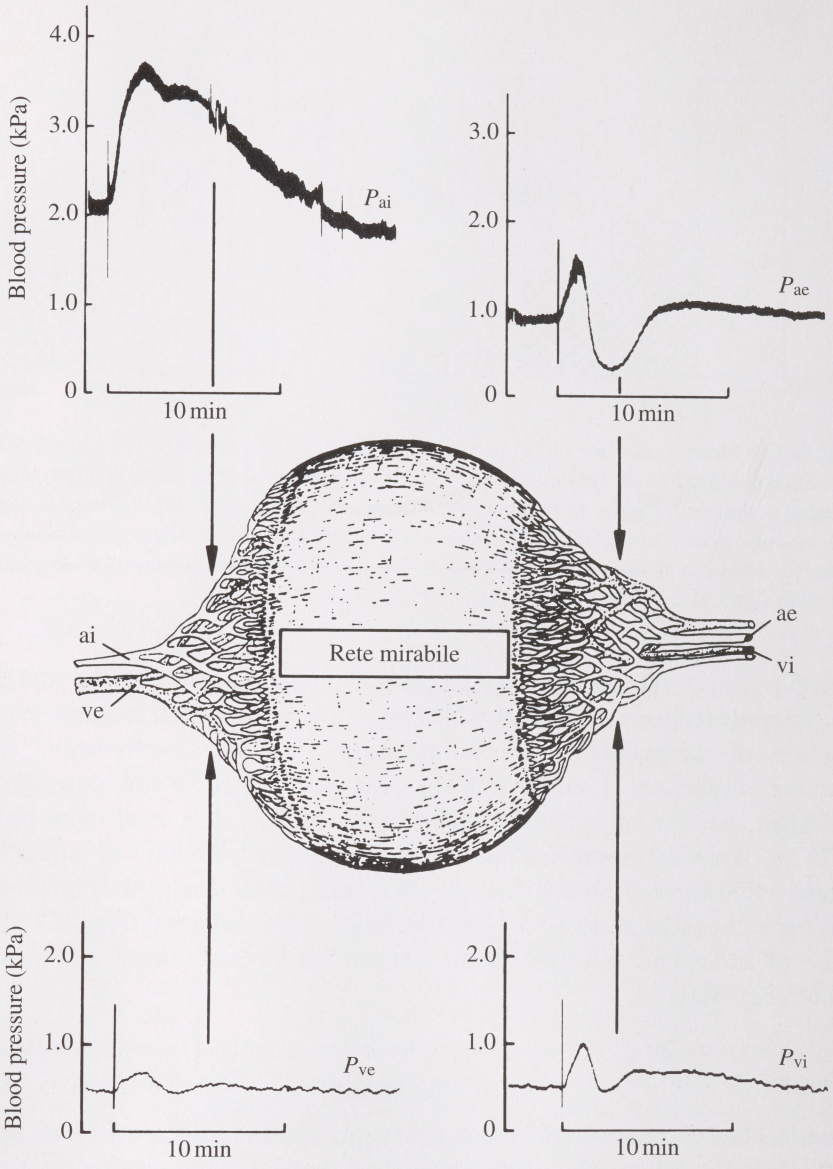

Fig. 5. Typical blood pressure traces measured in arterial and venous blood vessels proximal and distal to the rete mirabile during injections of adrenaline (4 nmol kg^{-1}) into the dorsal artery. (Recordings from various preparations.) ai, arterial influx; ae, arterial efflux; ve, venous efflux; vi, venous influx.

vessels of the rete mirabile is more than twice as high as the resistance of the venous vessels of the rete mirabile or of the capillary bed of the secretory swimbladder (Fig. 6B).

Mean changes in arterial blood pressure proximal and distal to the rete mirabile, in swimbladder blood flow and in the resistance of the arterial section of the rete mirabile following an injection of adrenaline into the dorsal artery are presented in Fig. 7.

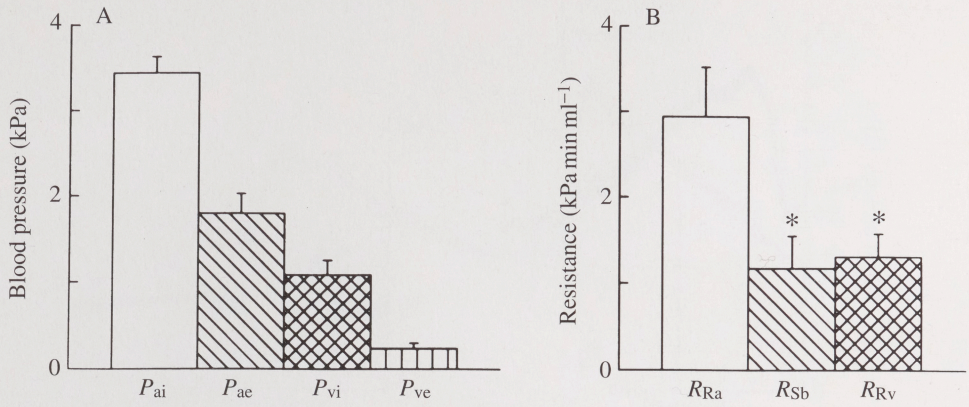

Fig. 6. (A) Mean values for blood pressure measured in arterial and venous blood vessels proximal and distal to the rete mirabile and (B) mean resistance of the three capillary systems present in the swimbladder circulation [Ra, arterial capillaries of the rete (between ai and ae); Sb, vascular system of the secretory bladder (between ae and vi); Rv, venous capillaries of the rete (between vi and ve)]; values are mean ± S.E.M., N=7. Asterisks indicate that R_{Sb} and R_{Rv} are both significantly different from R_{Ra}.

Adrenergic stimulation caused a marked increase in the resistance of the arterial part of the rete mirabile (between ai and ae), which only slowly returned to the preinjection level.

Injection of adrenaline after preincubation with the β-adrenergic antagonist propranolol resulted in a much smaller initial increase in blood flow through the swimbladder, but the subsequent decrease in blood flow was even more pronounced (Fig. 8A,B). When the adrenaline injection in the same preparation was repeated after an additional application of the α-adrenergic antagonist phentolamine, the initial increase as well as the subsequent decrease in blood perfusion were abolished (Fig. 8C). Increasing the dose of adrenaline, however, again induced the biphasic changes in swimbladder perfusion (Fig. 8D).

Discussion

Injection of adrenaline into the dorsal artery provoked an increase in blood pressure as well as in cardiac output. The relative changes in both variables were quite similar (see Fig. 3), suggesting that the observed changes mainly represent effects on the cardiac system rather than changes in total peripheral resistance. Similar changes in dorsal arterial blood pressure have been described for the European eel (Peyraud-Waitzenegger *et al.* 1980) and appear to be typical for teleosts as well as for elasmobranchs (Jones and Randall, 1978).

Adrenergic resistance vessels in the rete

The experiments with the blood-perfused swimbladder preparation provide strong evidence for the existence of α-adrenergically controlled resistance vessels in the swimbladder circulation. Application of a β-adrenergic agonist also caused a decrease in

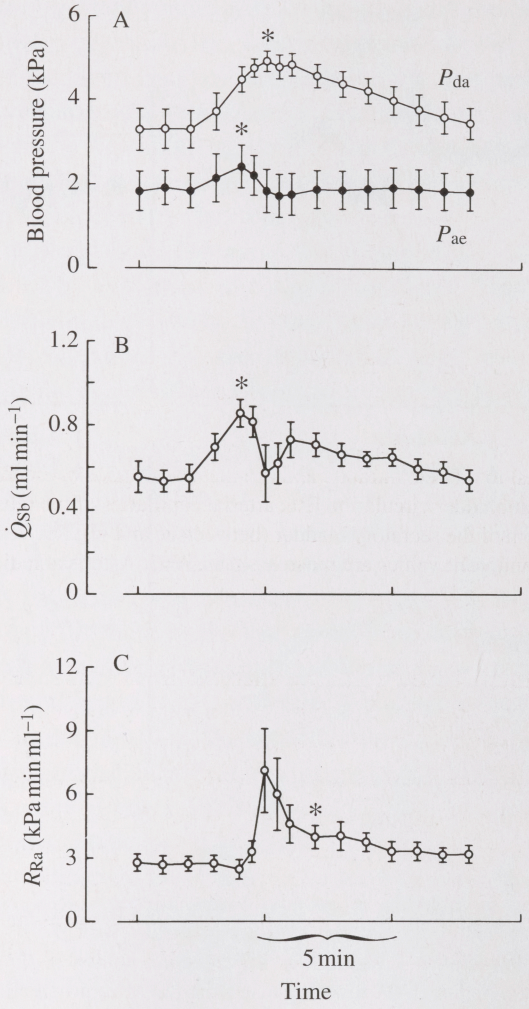

Fig. 7. Mean changes in blood pressure proximal (P_{da}) and distal (P_{ae}) to the rete mirabile (A), in swimbladder blood flow ($\dot{Q}_{Sb}$) (B) and in resistance of the arterial part of the rete mirabile (R_{Ra}) (C) following an injection of adrenaline (4 nmol kg^{-1}) into the dorsal aorta; values are mean ± S.E.M., N=5. Asterisks indicate significant differences compared with control values before injection of adrenaline.

perfusion pressure, while an appropriate antagonist caused an increase in perfusion pressure. In contrast to the α-adrenergic vasoconstriction, which was observed during each single injection of an α-adrenergic agonist, the β-effects were not very pronounced and were only observed in response to the first application of the drugs. The presence of α-adrenergically controlled resistance vessels has also been suggested by previous studies using saline-perfused swimbladder preparations (Stray-Pedersen, 1970; Nilsson, 1972; Wahlqvist, 1985).

The results of the *in situ* experiments of the present study demonstrate that these

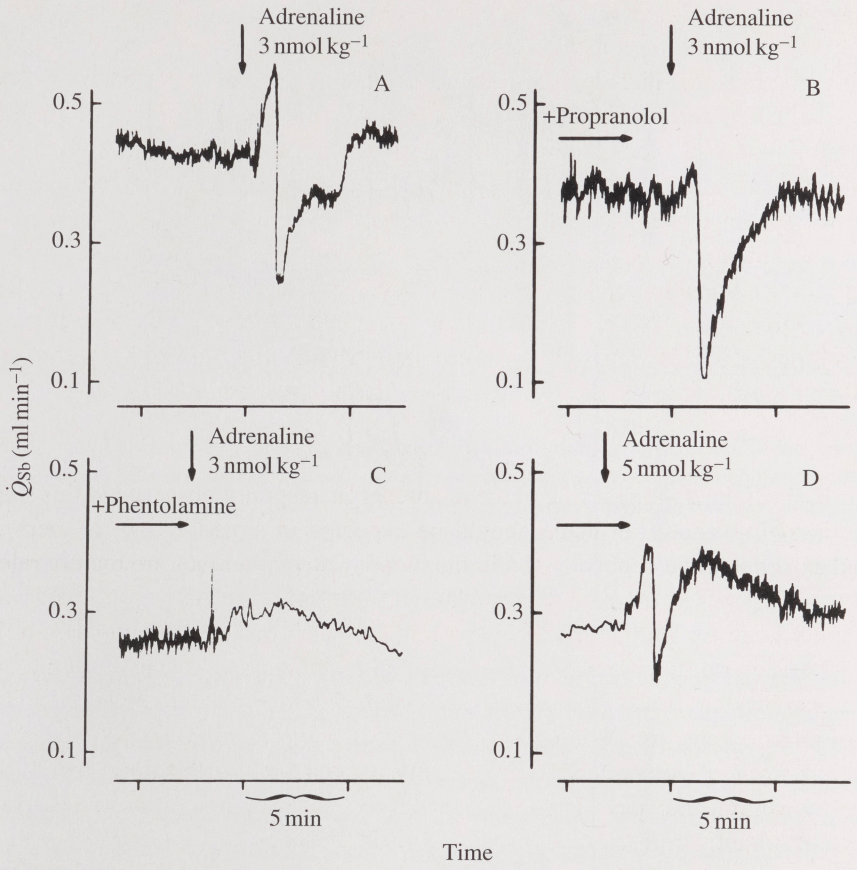

Fig. 8. Changes in swimbladder blood flow during injections of adrenaline into the dorsal aorta under control conditions (A) and after preincubation with β- (B) and α-adrenergic (C) antagonists. (D) Subsequent (20 min after the injection shown in C) injection of a higher concentration of adrenaline. (All recordings are from the same preparation.)

adrenergically controlled resistance vessels can indeed regulate swimbladder perfusion *in vivo*. The resistance of the swimbladder vascular bed increased markedly during application of catecholamines, causing a reduction in swimbladder blood flow in spite of the simultaneously elevated cardiac output. According to the pressure measurements proximal and distal to the bipolar rete mirabile, the resistance vessels must be located in the rete or very close to it. The most likely location appears to be at the arterial entrance of the rete mirabile. No evidence was found for the existence of additional resistance vessels or adrenergic control sites in the vascular bed of the secretory bladder (between ae and vi) or in the venous part of the rete mirabile (between vi and ve).

The biphasic response to adrenaline observed in swimbladder perfusion and in blood pressure in the vascular system distal to the rete suggests that there is both β-adrenergic vasodilation and α-adrenergic vasoconstriction. The increase in resistance coinciding with the decrease in blood flow, and the reduction of the effect after preincubation with

specific α-antagonists *in situ*, clearly indicates that there is α-adrenergic vasoconstriction. Although in this *in situ* preparation the α-adrenergic antagonist phentolamine will diminish the α-receptor-mediated changes in cardiac output, the perfusion experiments demonstrate that α-receptors in the swimbladder circulation do respond to adrenergic stimulation at constant blood flow. The absence of the decrease in swimbladder blood flow thus clearly indicates that α-adrenergic receptors in the swimbladder mediating a vasoconstriction have been blocked by phentolamine.

The initial increase in blood flow, preceding the α-adrenergic vasoconstriction was, however, not accompanied by a decrease in vascular resistance. Therefore, aside from a possible β-adrenergic vasodilatory effect, it could also be the result of the elevated cardiac output. This is supported by the observation that, even in the perfused swimbladder preparation, the β-adrenergic vasodilation was very weak.

Physiological significance

On the basis of the dominant role of the α-adrenergic response observed in this study, a decrease in swimbladder perfusion should be expected in situations where there is an increased catecholamine concentration in the blood. Catecholamines are mainly released from chromaffin tissue (Mazeaud and Mazeaud, 1981). A reduction in swimbladder blood flow will result in a decrease in the rate of gas deposition (Pelster and Scheid, 1992*b*). This suggests that the fish in a 'fight or flight' situation, when high concentrations of catecholamines are found in the blood, will shut down swimbladder perfusion to the benefit of other organs. In this situation, the swimbladder can no longer compensate for changes in buoyancy experienced during vertical movements. With filling rates of about one bladder volume per day (Fänge, 1983), gas deposition is a slow process and is certainly not suitable for instantaneous adaptations.

Another aspect is the mechanism provoking an increase in swimbladder perfusion and thus a stimulation of gas deposition. The presence of β-adrenergic receptors causing vasodilation suggests that a selective stimulation of these receptors could be involved. The observed β-effect, however, was not very pronounced and thus an increase in blood flow could also be the result of a reduction in the α-adrenergic tone. Swimbladder blood vessels are innervated by adrenergic nerves because electrical stimulation of the splanchnicus or the vagosympathetic trunk causes a vasoconstriction of cod swimbladder vessels and both nerves stain for catecholamines (Nilsson, 1972; McLean and Nilsson, 1981; Wahlqvist, 1985). In these studies, the vasoactive blood vessels could not be localized. In the present study, blood-borne stimuli were used to identify adrenergic resistance vessels in the eel swimbladder, and it would be interesting to know whether the vessels reached by catecholamines from the blood also are under nervous control.

Several additional vasodilatory mediators have been described, and vasoactive intestinal peptide (VIP), a potent vasodilator, has been found in the cod swimbladder (Lundin and Holmgren, 1984). Somewhat surprising, however, was the observation of Lundin and Holmgren (1991) that VIP caused a reduction in the secretory activity in cod swimbladder, because a vasodilation usually coincides with a high secretory activity. This effect could be the result of a modification of the cholinergic tone by VIP. The swimbladder is obviously equipped with a number of different mechanisms that cause a

vasodilation, but none of them appears to dominate. How vasodilation is actually achieved *in vivo* still remains to be determined.

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